was a case of typhoid fever with a complicating cholecystitis. The case is here reported because the disease was watched, treated medically, and operated upon without the slightest suspicion of a typhoidal origin.

CASE X.—On November 3, 1897, Mrs. S., aged sixty-seven years, a patient of Dr. Gay, was taken with severe pain in the epigastrium extending toward the left. This attack had been preceded by a few days of uneasiness in the region of the stomach. The temperature was 102°. There were vomiting and distention.

The patient had been subject to "bilious" attacks, but had never been jaundiced. The present attack was supposed to be indigestion, and little was thought of it. The next day the pain was much more severe, and was localized in the region of the gall-bladder. There was constipation of gas and feces. There were general distention of the abdomen, tenderness over the gall-bladder, and dulness in the flanks. The diagnosis of acute cholecystitis was made. On the evening of the second day the temperature had fallen to 100°, the pulse to 90—the patient was decidedly better. Operation was therefore deferred.

On the following day the pain increased in severity; the pulse and temperature rose; the abdominal symptoms were more acute. Both Dr. Gay and myself concluded that interference was imperative, and I was asked to operate, Dr. Gay being called away unexpectedly.

An extremely distended gall-bladder was found, everywhere adherent by recent exudate. It was dark in color, and contained numerous calculi suspended in a dark, hemorrhagic, turbid fluid. The mucous membrane was dark gray and œdematous. The gall-bladder was drained with tube and gauze. Fully as many gallstones escaped through the tube as were extracted at the time of operation. The patient made a good recovery, and remains well. Cultures taken from the gall-bladder showed numerous bacteria of different kinds, which were supposed to be contaminations, so that no conclusions could be drawn as to the nature of the infection in this case.

THE CLINICAL SIGNIFICANCE OF REDUPLICATION OF THE HEART-SOUNDS.

By HENRY SEWALL, PH.D., M.D.,

PROFESSOR OF PHYSIOLOGY IN THE UNIVERSITY OF DENVER; VISITING PHYSICIAN TO ST. LUKE'S AND ARAPAHOE COUNTY HOSPITALS, DENVER.

THE general fact that the two sounds of the heart may, either one or both, be heard as double sounds, is referred to in all works upon physical diagnosis. This incomplete summary of the opinions of different observers is only introduced to show that there is no settled belief as to the facts of reduplication, whether as to the frequency of the phenomenon, its clinical significance, or its conditions of occurrence. Eichhorst[1] states that reduplication of the heart-sounds is rare. According to Guttmann,[2]

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[1] Eichhorst. Physikal. Untersuchungsmethoden, 1889, vol ii. p. 54.

[2] Guttmann. Handbook of Physical Diagnosis. Translated by Napier, 1880, p. 210.

"Division of the first ventricular sound is relatively most frequent, the next most common phenomenon of this kind being doubling of the second arterial sound. Reduplication of the first ventricular sound occurs in perfectly healthy people, but is then never permanent."

Rosenstein[1] states that ventricular reduplication is rare. Osler[2] says "Gallop rhythm . . . usually results from the reduplication of the second sound in a rapidly acting heart. . . . Sometimes it seems as if the first sound was split; more commonly it is the second." Most authors mingle a theory of causation with the statement of the fact of reduplication. Thus, Bristowe[3] writes, "Asynchronism in the action of the ventricles, indicated by reduplication of the sounds of the heart, is not infrequently observed. Occasionally it occurs in health, but much more commonly it is an accompaniment of disease. Reduplication of the first sound is chiefly met with in connection with hypertrophy of the heart and high arterial tension, especially, therefore, in chronic heart disease; and reduplication of the second sound is observed mainly in affections of the mitral valve." According to Fagge and Pye-Smith,[4] "If the tension between the two great vessels of exit from the heart is very different in degree, the pulmonary valves may close before or after those of the aorta, and thus the second sound becomes reduplicated. Reduplication of the first sound at the apex undoubtedly occurs, but in a well-marked and unmistakable form is decidedly rare." Flint[5] writes, "Reduplication of the heart-sounds denotes a variety of functional disorder giving rise, however, to no definite subjective symptoms, and occurring in various pathological conditions . . . a slight deviation from synchronism in contraction of the right and left ventricle accounts satisfactorily for the reduplication of both sounds, but not so satisfactorily for a reduplication limited to the second sound. The latter is best explained by supposing that, under certain conditions of arterial pressure, the recoil of the aorta and of the pulmonary artery after the ventricular systole is not in unison." From the physiological side Foster[6] may be quoted, "In certain cases in which the semilunar valves on the two sides of the heart are not wholly synchronous in action, the sound of the heart here (at the second right costal cartilage) is double ('reduplicated second sound'), one being due to the aorta and one to the pulmonary artery." Some views are interesting chiefly as examples of the readiness with which explanations of clinical phenomena may be framed; thus, Barclay[7] writes, "Reduplication, like intermission, suggests some imperfection in the relation of nervous force and muscular contraction. . . . Reduplication of the second sound is very often caused by imperfect closure of the auriculo-ventricular aperture on one side, which causes the systole of one ventricle to terminate more quickly than the other." Sahli[1] states, "A splitting of the first sound must depend on non-synchronous beginning, and of the second sound on non-synchronous ending of the systole of the two ventricles. The frequent explanation of the non-synchronous closure of the semilunar valves (as causing reduplication of the second sound) is undoubtedly wrong." Vierordt[2] shows the confusion arising from attempted comparison of physiological with pathological conditions. "Divided or double heart-sounds are ordinarily without significance if the condition otherwise is one of health. . . . Division of the second sound at the apex occurs in mitral stenosis. . . . We may especially refer a divided second sound at the apex, according to my experience, to mitral stenosis, in case there are, besides, undoubted signs of mitral insufficiency. Further, a divided second sound is heard in *pericarditis adhesiva* and systolic retraction of the apex beat." Sahli,[3] on the other hand, is one of the few observers who correctly state that the triple rhythm heard at the apex in mitral stenosis is not caused by reduplication of the second sound. Walshe[4] made an exhaustive study of this subject; but, as will be made clear later, he apparently confused real with pseudo-reduplication of the heart-sounds.

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[1] Rosenstein. Ziemssen's Cyclop. Pract. Med., vol. vi. p. 52.

[2] Osler. Practice of Medicine, 1892, p. 651.

[3] Bristowe. Practice of Medicine, 1890, p. 516.

[4] Fagge and Pye-Smith. Principles and Practice of Medicine, 1891, vol. ii. p. 63.

[5] Flint. Practice of Medicine, 1886, p. 353.

[6] Foster. Text-book of Physiology, 1893, vol. i. p. 239.

[7] Barclay. Manual of Medical Diagnosis, 1864, p. 292.

Gerhardt[5] states truly that the phenomenon of division of the heart-sounds can be attributed to very different causes. He cites cases in which clinical examination disclosed reduplicate heart-sounds over limited areas, and in which, examined post mortem, there were found tendinous patches on the heart, confined to the areas of reduplication, whose friction had caused the phenomenon. Among the most important researches on this subject are those of Potain[6] and of Geigel.[7] Their original compositions have not been accessible to me. The former author found one or both heart-sounds to be doubled in one-fifth of all cases investigated, and usually it was the first sound that was split. He remarked that, in any case, the reduplication tended to rhythmically come and go with a period dependent on the rate of respiration. He observed that the doubling of the first sound was most marked at the end of expiration and beginning of inspiration, while the widest splitting of the second sound occurred at the end of inspiration and beginning of expiration. Of the divided first sound, the first half was heard more strongly over the left ventricle and the second half over the right ventricle. Of the divided second sound, the first half was heard more loudly in the aortic area, right of the sternum, and the second half over the pulmonary artery. The conclusion was, of course, that the contraction of the left ventricle and the closure of the aortic valves, in cases of reduplication, preceded the similar events on the right side of the heart. Geigel pointed out that changes in the ratio of pulmonary to aortic blood-pressure could alter the rate of arterial diastolic rebound so as to cause a perceptible interval between the closures of the two sets of semilunar valves. The opinion of W. H. Broadbent[1] has particular value: "Under certain conditions, chiefly when there is hypertrophy and dilatation of the left ventricle as a result of renal disease, or, on the other hand, when the right ventricle is overtaxed by resistance in the pulmonary circulation, the two ventricular sounds are not absolutely synchronous. When this is the case, the first sound as heard, either over the right ventricle or just outside the apex, is prolonged or murmurish, as it was termed by the late Dr. Sibson; but when the stethoscope is planted a little to the inner side of the apex the confused sound resolves itself into two distinct elements, and is found to be duplex or reduplicated. That is, the right and left sounds are heard separately, and the reasons why they are best so heard at the point mentioned is that here the stethoscope is over the septum, between the ventricles and upon a small part of each, so that it conveys the sound from both. . . . Reduplication of the second sound from precipitate or retarded closure of pulmonary or aortic semilunar valves disturbing the normal synchronism between the two is a frequent effect of mitral stenosis and of pericarditis, and it may be met with as a result of adherent pericardium and of other structural or valvular lesions. It is rarely produced by systemic resistance and high arterial tension, which give rise to reduplication of the first sound; more frequently by high tension in the pulmonary circulation due to disease of the lungs. It is, in effect, the pulmonary sound which is displaced, as is, perhaps, sufficiently shown by the fact that reduplication can be induced by simply holding the breath." In an able discussion of the subject, George Johnson[2] expresses still other views: "I now venture to suggest that the true explanation (of reduplication of the first sound) is to be found in the fact that the contraction of a dilated, and especially of an hypertrophied, auricle becomes sonorous, and that the first division of the double first sound in the cases under consideration (cases of Bright's disease) is the result of the auricular systole. . . . Dr. Hayden, in his recently published and valuable work on *Diseases of the Heart and Aorta*, expresses the opinion that reduplication of the first sound 'is due to resolution of the first sound into its two normal elements, namely, the cardiac impulse and the sudden tension of the auriculo-ventricular valves. . . . Cardiac impulse coincides with the initial portion of the ventricular systole, while tension of the auriculo-ventricular valves and attached *chordæ tendineæ* occurs at the conclusion or acme of the systole.'" Hayden,[1] drawing his conclusions from a carefully studied case in which there was aortic stenosis, together with both mitral stenosis and incompetency, and in which doubling of the second sound was very marked, held that the first element of the reduplicated second sound was caused by closure of the semilunar valves; but that the second element depended upon the sudden tension of the mitral curtains by reflux of the sharp current sent through the centre of the ventricle by the resilience of the hypertrophied auricle, and which passes back along the walls under the valves. If this valve-tension occur late in diastole it would simulate doubling of the first sound. Barr[2] explained reduplication as the physiological result of the rhythmic over-filling of the right ventricle which, he says, normally occurs at the end of expiration and beginning of inspiration. The right ventricle is thus stimulated to contract before the left, the result being a doubling of the first sound. As pulmonary capacity increases with inspiration, the resistance to contraction of the right ventricle is decreased, its systole is abbreviated, and the closure of the pulmonic valves is hastened. Barr thus supposes the conditions leading to reduplication to be initiated in the right ventricle, while most authors ascribe this phenomenon to occurrences in the left ventricle. Finally, Sansom[3] has elaborately reviewed the whole subject. It is a great merit on the part of this author to have distinguished *real* from *simulated* reduplication of the heart-sounds. He says, in part: "What seems a doubling of either of the sounds is really a complex phenomenon dependent on various causes. The sounds which so closely resemble, or may be to the ear absolutely indistinguishable from, doublings of the sounds, are not necessarily repetitions of the systole of the ventricles, or of the reflux against the semilunar valves, but may be due to many causes, and may occupy other portions of the cardiac cycle than those which have just been indicated." Sansom concludes from his studies that the so-called reduplication of the first sound of the heart is not due to want of synchronism in the action of the two ventricles, but to audible, presystolic vibration of the mitral valve which is produced by the sudden tension imparted to it by reflux of the current shot into the ventricle by the contraction of the (usually hypertrophied) auricle. The first element of the double sound is, then, presystolic and valvular. The same explanation is offered for reduplication of the second sound, except that the mitral valve tension is produced early in diastole, just following semilunar closure, by the reflux of a current sent into the ventricle by the elastic resilience of an over-stretched auricle. These views differ from those previously announced by Hayden, inasmuch as, according to this author, it is the *second* element of the double first sound which is valvular in origin.

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[1] Sahli. Lehrb. d. klin. Untersuchungsmethoden, 1894, S. 221.

[2] Vierordt. Medical Diagnosis. Translated by Stuart, 1894, p. 219.

[3] Sahli. Lehrb. d klin. Untersuchungsmethoden, 1894, S. 221.

[4] Walshe. Diseases of the Heart, 1862, pp. 73-76.

[5] Gerhardt. Lehrb. d. Auscultation u. Percussion, 1890. S. 210

[6] Potain. Note sur les dedoublements normaux des bruits du cœur. Union Médicale, 1866, vol. xxxi. (Quoted by Gerhardt.)

[7] Geigel. Die gespaltene Herztöne. Memorabilien. Heilbr., 1868, vol. xiii. Verhandl. d phys. med. Gesellsch. in Wurzburg, 1869, vol. i (Gerhardt.)

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[1] W. H. Broadbent. On Some Points in Relation to the Sounds of the Heart. Practitioner, vol. xxx. p. 1.

[2] George Johnson. *Lancet*, 1876, vol. i. p. 699.

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[1] Hayden. (Quoted by Sansom, p. 216.)

[2] Barr. Medical Times and Gazette, 1877, vol. i.

[3] Sansom. Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892, p. 207.

It must now be clear that there is little harmony in the various interpretations of the phenomena of reduplication of the sounds of the heart. The scientific character of those who have already investigated this subject is sufficient evidence of its importance, and the unsatisfactory condition of our present knowledge is reason enough why further effort should be made to explain our clinical facts.

(To be continued.)

HÆMATOMYELIA FROM GUNSHOT WOUNDS OF THE SPINE.

A REPORT OF TWO CASES, WITH RECOVERY FOLLOWING SYMPTOMS OF HEMILESION OF THE CORD.

By Harvey W. Cushing, M.D.,
RESIDENT SURGEON OF THE JOHNS HOPKINS HOSPITAL.

Gunshot injuries of the spinal column are of comparative rarity. The mortality of cases with symptoms of cord lesion, especially in the cervical region and without operative interference, reaches a high percentage.[1]

In those cases which recover, the nature of the lesion is often difficult of determination, the disturbances of motility and sensibility giving clue alone to the spinal segments involved and the transverse extent of the injury, but not to its pathological anatomy.

In each of the following cases the missile lodged in the centrum of a vertebra without direct injury to the cord; but, nevertheless, produced symptoms of hemilesion. A study of their symptomatology, with a discussion of the character of the lesion, is contained in this communication.

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[1] In the American Civil War 642 cases of gunshot wound of the spine of all varieties were reported, with a fatality of 55 per cent. The cervical cases alone reached 70 per cent. Chipault (Etudes de Chirurgie Médullaire. Paris, 1894) has collected 104 cases from 1850 to 1891, with operation and recovery in 60 per cent. The mortality without operative interference reaches 80 per cent., and life, for the 20 per cent. surviving, was hardly worth the living. The consensus of opinion (cf. Vincent, Revue de Chirurg., 1892) recommends operation unless there is total transverse lesion indicated by abolition of knee-jerks.